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DIAGNOSIS AND NO. OF PATIENTS	DURATION OF TREATMENT	RESULTS	ADVERSE REACTIONS
Impetigo contagiosa (50)	Triburon 3-10 days	excellent	none
Ecthyma (24)	Triburon 5-14 days	excellent	none
Pustular folliculitis (16)	Triburon 1-3 weeks	infection controlled	3 complaints of burning
Dermatitis repens (18)	Triburon 1-2 weeks	infection controlled	none
Dermatitis repens (18)	Triburon-HC 1-4 weeks	excellent	none
Secondarily infected eczematous eruption (20)	Triburon 1-2 weeks	infection controlled	none
Secondarily infected eczematous eruption (64)	Triburon-HC 1-4 weeks	excellent	1 complaint of irritation

Chart based on finding of E. Edelson, E. Grunberg and T.V. Morton, Jr.

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Triburon-HC Ointment, 5-Gm and 20-Gm tubes.

REFERENCES: 1. R. J. Schnitzer, E. Grunberg, W. F. DeLorenzo and R. E. Bagdon, to be published.
2. E. Edelson, E. Grunberg and T. V. Morton, Jr., Antibiotics Annual 1958-1959, New York, Medical Encyclopedia, Inc., 1959.
3. R. C. V. Robinson and L. E. Harmon, Antibiotics Annual 1958-1959.

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SOUTHWESTERN MEDICINE

VOL. XL MAY, 1959 No. 5

Hypotensive Drugs

By LESTER C. FEENER, M.D., F.A.C.P., EL PASO

(CONTINUED)

For the sake of completeness, the antiserotonins must be mentioned. It is felt that perhaps the antihypertensive effect of Reserpine is related to its effect on the serotonin body stones. Some investigators believe that it actually depletes the body of serotonin. Serotonin is under investigation in a number of diseases, and essential hypertension is one of them.

An agent called BAS has been used in the treatment of hypertension. It has been discontinued in most cases, mainly because the side-effects of excessive sedation have limited its use. However, in the same connection, Iproniazid has been used in hypertension. This however is strictly not antiserotonin. It may be better called a "proserotonin."

However, we do know that when this drug is added to certain cases of hypertension, which are resistant to all drugs including chlorothiazide, this drug has reduced blood pressure. We do not, however, know what its action is. We do know that it can produce postural hypertension, and it may cause serious liver damage. For that reason it should be considered as a purely experimental drug at the present time in the treatment of hypertension.

Headache Plus Convulsions

The parental use of antihypertensive drugs is probably limited to severe hypertension with headache plus convulsions. The drug most frequently used is intramuscular Reserpine in a dose of 1 to 2.5 mgs several times daily, checking the blood pressure frequently. To this may be added intramuscular magnesium sulphate. (two cc of 50 per cent solution) every few hours.

This combination of drugs has usually been sufficiently effective. However, if it is felt that an emergency exists, where a speedy reduction is urgently required, then intravenous or intramuscular blocking agents may be used. It must be remembered that the use of these drugs is fraught with danger, and they must be supervised very carefully. The drug usually used is hexamethonium.

Perhaps the safest way to use it is by the subcutaneous or intramuscular route, giving not more than five mgs every 15 minutes until the desired result is obtained. Other workers have used this blocking agent in one mg doses intravenously every three to five minutes.

It is repeated that the use of these drugs must be carefully supervised, and usually this type of urgent therapy is not required, and the patient does very well on intramuscular Reserpine with magnesium sulphate plus sedation.

Lumbodorsal Sympathectomy

Since the advent of antihypertensive drugs, lum-

CURRENT THERAPY

* Dr. Feener this month is substituting for Dr. Jack A. Bernard, regular condustor of this section.

bodorsal sympathectomy has not been used to the extent that it had been previously. However, it must be remembered that a young hypertensive who has the disease severely may not be able to be adequately controlled, upon drug therapy alone.

In the past few months, there is a growing tendency to feel that one might do a less extensive operation, and then add in the antihypertensive therapy, and this would be an effective means of controlling this type of case over a period of years.

There is not any large series of cases and for the present one must evaluate his young hypertensive and if sympathectomy is to be considered probably an adequate type of operation should be done.

It is entirely possible if one is careful to add a hypotensive regime to an adequately sympathectomized patient should this become necessary after a period of years.

Diet in Hypertension

In closing, a few words should be said about diet in the treatment of hypertension. Since the advent of the drugs, especially since the use of chlorothiazide, dietary regimes have not been considered as necessary. However, it will be found that certain cases of severe hypertension with kidney damage plus nitrogen retention will still require dietary therapy, and these cases will require as stringent a diet as the so-called rice-fruit-sugar diet which is essentially a low protein, low sodium regime. Hypertension may exist with the collagen diseases, such as periarteritis nodosa and disseminated lupus. Hypertension is simply part of the disease, and will respond to steroid therapy.

404 Banner Building

"Before and After" Atherosclerosis

"Many patients operated upon for aneurysms and occlusive disease are in the later decades of life and have a relatively short life expectancy. Thus, the time from operation to death is an opportunity to investigate certain aspects of lipid metabolism as it relates to atherosclerosis. At operation, the diagnosis of atherosclerosis could be made with certainty and the type and extent of the atheromatous lesions recorded. Subsequently, at necropsy, whether months or years later, the arterial system could be re-examined and the findings compared with those obtained previously. Thus, a series of "before and after" observations would be available against which factors such as the natural history of the disease, metabolic alterations, and effectiveness of drug therapy could be evaluated. To a surgeon, at least, this sort of investigation is more appealing than one in which the diagnosis is based solely upon clinical manifestations, the electrocardiogram, the blood lipids, and in which the same indirect criteria are employed in evaluating results."

—OSCAR CREECH, Jr., M. D.
in *American Heart Journal*
p. 642, Nov. 1957

W. Compere Basom, M.D., *El Paso, Orthopaedic Editor*

ORTHOPAEDIC SURGERY NOTES

The Orthopaedic Editor Views THE PRACTITIONER

Sir Heneage Ogilvie, the editor of THE PRACTITIONER published in London very kindly gave this editor permission to abstract orthopaedic information from this excellent publication.

The August 1958 issue is concerned with industrial medicine.

This journal contains an article each month entitled "Motoring Notes" and also an item concerning "Travel Notes". These are very interesting subjects.

'Notes From The Continent" touch on some of the latest developments from the European Countries. "Notes Concerning Preparations" give information on the newer medications and medical news of Great Britain.

The industrial medicine issue contains an article concerning "Sprains" by F. C. Derwin, orthopaedic surgeon (Elizabeth Orthopaedic Hospital, Exeter).

Ligaments and Muscles

In this, the synovia, ligament and muscle attachment damage is discussed. Even though the radiological examination may be negative, it is important that the condition not be necessarily regarded as trivial or unimportant.

Joints like the knee and ankle are mechanically unstable. These joints depend on their ligaments and muscle control for stability. If the muscles are inadequate, then they give way and the strain effects fall on the ligaments.

These may be stretched or torn or the attachment of the ligament to the bone may be torn off bringing with it a small flake of bone.

The ligament can be ruptured completely. Complete tears of the ligaments are uncommon but when they occur the rupture seldom takes place in the middle of the ligament. It is nearly always situated at the proximal or distal end. It is important to recognize this early, as in some instances operative repair is required.

Swelling of the joint is due to out-pouring of serofibrinous exudate occurring as an immmediate synovial response to the injury. If the swelling occurs within a few minutes after the injury then usually it is a hemarthrosis following rupture of blood vessels.

Sometimes this exudate undergoes organization leading to the formation of fibrous bands or adhesions and this may produce long standing disability of pain, restricted movement and swelling.

With a complete tear there is usually instability before pain occurs on testing and in case of doubt it is well to examine the joint under anesthesia and to combine this with radiological examination.

It is most important that the treating physician insist on a radiographic examination in case of injury even though it appeared trivial and that radiography is unnecessary. It is also essential that special views be taken in order to disclose certain occult fractures, notably the fracture of the carpal os scaphoideum.

Also, it is well to remember that occasionally a fracture of this bone will show up several weeks later whereas a hairline fracture may not even be discernible by radiographic examination early. "So that it is wise to repeat the radiographs in one to two weeks if the first are negative but the fracture may seem likely" (Editor's opinion).

Treatment. Cold compresses, elevation and rest support for relief of swelling, contrast baths and whirlpool baths can be used. Plaster support should be used if there is any doubt or question as to ligament rupture. Complete recovery from most simple sprain should take place in 3 to 4 weeks.

With chronic sprains it may be necessary to do a manipulation under anesthesia to free up the adhesions which are restricting motion and causing it to be painful. It is also a good idea to re-x-ray the area again prior to the manipulation.

Editor's Note: Actually all practitioners are specialists in that there are conditions which they do treat and conditions which they don't. Every specialist must be a general practitioner in that the human body cannot be divided up into areas and treated solely with regard to the area. The patient must be treated as an entire entity and therefore the average specialist must be a general practitioner at least in the diagnostic field.

It is therefore, always of interest to this reviewer to read some of the general medical publications, and "THE PRACTITIONER" is most interesting.

Dr. Blasingame

MEETINGS

Biological-Astronomical Space Missile Expert To Speak at New Mexico Medical Society Meeting

Dr. Eugene B. Konecci of Douglas Aircraft in Santa Monica, head of the Department on Human Factors in Biological-Astronomical Missiles in Space Travel, has been added to the group of outstanding speakers who will address the 77th annual meeting of the New Mexico Medical Society at Las Cruces, N. M., on the subject of space medicine, May 5-7.

Dr. Konecci will act as moderator of a panel of experts, who will discuss "Atmospheres for Space Travel."

Present will be Dr. F. J. L. Blasingame, Chicago, Executive Vice President of the American Medical Association and Past President of the Texas Medical Association. His roundup on AMA activities is scheduled for the noon luncheon in Milton Hall May 6.

Other Panels

Other panels will be moderated by Col. John P. Stapp, MC, Chief, Aeromedical Laboratory at Wright Air Development Center, Dayton, Ohio; Dr. Randolph W. Lovelace of the Lovelace Clinic in Albuquerque, chairman, National Aeronautics and Space Administration Special Advisory Committee on Life Sciences; Col. John C. Pickering, MC, Randolph Air Force Base, San Antonio, Chief of Research, School of Aviation Medicine; Capt. George Ruff, MC, Chief, Biophysics Branch, Aeromedical Laboratory at Wright Air Development Center, Dayton, Ohio; and Hebertus Strughold, M.D., Ph. D., Randolph Air Force Base, San Antonio, Professor of Space Medicine and Adviser for Research at Headquarters School for Aviation Medicine.

Experts in the field of space medicine throughout the U. S. will participate in the panels. Availability of several was not expected to be known until convention time.

Tour of Holloman

The panels will be held on May 6 and 7 in Milton Hall of New Mexico State University in Las Cruces. On the preceding day physicians attending the convention will go on a tour of Holloman Air Force Base, a few miles from Alamogordo, where the Air Force Missile Development Center of the Air Research and Development

Command is located. The program may include a high speed rocket sled run on the new 35,000 foot captive missile test track and a MACE or Aerobee launching.

In announcing the final program, Dr. James C. Sedgwick expressed appreciation to Lt. Col. Rufus R. Hessberg, MC, Chief, Aeromedical Field Laboratory, Air Force Missile Development Center at Holloman Air Force Base, and his staff for arranging the complete scientific program for the space medicine meeting.

Registration

Registration will start at 7:30 a.m. May 5 in Milton Hall for those who do not plan to take the Holloman tour that day. At 8:30 a.m. buses will leave Milton Hall for the Holloman tour and return at 5 p.m. There will be a buffet and unstylish style show at the Las Cruces Country Club starting at 6:30 p.m.

The annual dinner dance will be held in the country club at 6:30 p.m. May 6.

Officers of the New Mexico Medical Society are:

Dr. James C. Sedgwick, Las Cruces, President; Dr. Lewis M. Overton, Albuquerque, President-Elect; Dr. Allan L. Haynes, Clovis, Vice President; Dr. Omar Legant, Albuquerque, Secretary-Treasurer; and Dr. Samuel R. Ziegler, Espanola, Immediate Past President.

Committee Chairmen

Convention Committee Chairmen, all of Las Cruces, are:

Dr. James C. Sedgwick, Convention Coordinator; Dr. Andrew M. Babey and Dr. Charles L. Harris, General Chairmen; Dr. A. Daniel Maddox and Dr. Ezra K. Neidich, Exhibits; Dr. William D. Sedgwick, Scientific Program; Dr. Remo E. Gay, Publicity; Dr. Arthur J. Fischer, Invited Guests.

Dr. Leland S. Evans and Dr. George Goossen, Entertainment; Dr. James F. McDowell and Dr. Carl Tarlowski, Housing and Transportation; Dr. Charles W. Carroll and Dr. John S. Drake, Specialty Luncheons.

Mrs. Leland S. Evans is in charge of registration and arrangements for the Women's Auxiliary

Specialty Meetings

Wednesday, May 6

7:30 A.M. Breakfast, New Mexico Pediatric Society, Milton Hall

Thursday, May 7

12:15 P.M. Luncheon, New Mexico Society of Internal Medicine, Annex Room, Town and Country

12:15 P.M. Luncheon, New Mexico Chapter, Western Orthopaedic Association, Maple Room, Town and Country. Guest Speaker: G. W. N. Eggers, M.D., Professor of Orthopaedics, University of Texas; "Experimental Immobilization and Remobilization of Joints." There will also be a panel discussion on surgery in cerebral palsy.

Dr. Lovelace *Col. Pickering* *Capt. Ruff* *Col. Stapp*

Friday, May 8

New Mexico Heart Association
Milton Hall

8:45 A.M. Registration

9:15 A.M. Welcome
Alan L. Frankel, M.D., Albuquerque, President, New Mexico Heart Association

9:20 A.M. Outline of Program
Andrew M. Babey, M.D., Las Cruces, Chairman, Presiding

9:25 A.M. "Some Shortcomings of the Electrocardiogram in the Diagnosis of Myocardial Infarction," Professor Demetrio Sodi-Pallares, M.D., Mexico City

10:10 A.M. Discussion

10:30 A.M. Coffee Break

10:45 A.M. "Atypical Rheumatic Fever," Arthur Moss, M.D., Los Angeles, Calif.

11:30 A.M. Discussion

12:00 Noon Luncheon

1:15 P.M. "Anti-Coagulant Therapy in Private Practice," Charles Stephens, Jr., M.D., Tucson, Ariz.

2:00 P.M. Discussion

2:20 P.M. "The Bedside Diagnosis of Some Common Congenital Heart Lesions," James K. Conrad, M.D., Albuquerque

3:00 P.M. Discussion

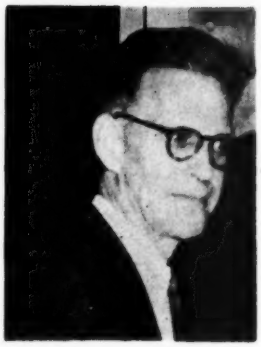
3:30 P.M. Adjournment

Technical Exhibits

Abbott Laboratories
Albuquerque Typewriter Exchange
Allied Medical Supply
A. S. Aloe & Company
Ayerst Laboratories
Ciba Pharmaceutical Products, Inc.
Coca-Cola Company
Eaton Laboratories
Eli Lilly and Company
Encyclopedia Britannica
Esco Bio-Chemical Company
Exercycle of New Mexico
Geigy Pharmaceuticals
General Electric Company
Lederle Laboratories
Mead Johnson & Company
Merck, Sharp & Dohme
Wm. S. Merrill Company
New Mexico Pharmacal Co.
New Mexico Physicians' Service
Organon, Inc.
Parke, Davis & Company
Pfizer Laboratories
Professional Men's Association
Department of Public Health, Committee on Aging
A. H. Robins Co., Inc.
J. B. Roerig & Company
Wm. H. Rorer
Ross Laboratories
Sandoz Pharmacal Co.
Schering Corporation
G. D. Searle & Company
Southwestern Surgical Supply Co.
E. R. Squibb & Sons
Upjohn Company
U. S. Vitamin Corp.
Warner Chilcott
Winthrop Laboratories

Social Security Footnote . . .

"A dangerous thing about Social Security in the United States," said Ray D. Murphy, president of Equitable Society, "is that the American people have not yet come to realize that more can be given only by taking more. The nation simply does not get something for nothing in Social Security."



Dr. Babey

Edited By ANDREW M. BABEY, M.D., LAS CRUCES, N.M.

APHORISMS and MEMORABILIA

Miscellaneous Truths and Concepts

1. ". . . extreme abdominal distension, a symptom to which I wish to call your attention as a complication of fractured rib."—RALPH BETTMAN, *Surgical Clinics, North America,* June 1935, p. 656.

2. "Purpuric spots are exceedingly rare in typhoid fever."—FRENCH, *Differential Diagnosis,* p. 670.

3. "Nothing predisposes so much to an attack of erysipelas as the fact that the patient has suffered from the disease previously."—KER, *Infectious Diseases,* 1929, p. 463.

4. "I should say about one case of typhoid in five gets diarrhea."—R. CABOT, *Clinical Conference,* 1932.

5. "Pea sized glands mean nothing. Half the people in the world have them. Grape sized—no. That means pathology of some kind"—R. CABOT, *New England J. Med.,* 204: 507, 1931.

Chicken-Pox

6. "Of all the eruptive fevers, chicken-pox is perhaps the least likely to occur twice in the same person."—KER, *Infectious Diseases,* 1929, p. 216.

7. "(Rose Spots)—are seen sometimes in cerebrospinal meningitis, typhus fever, trichinosis and acute miliary tuberculosis."—L. BARKER, *Internat. Clin.,* Vol. 2, 27th series, p. 32.

8. "When we have a fast pulse throughout in typhoid, we should turn our attention to the thyroid gland, for tachycardia in uncomplicated typhoid fever does not occur."—L. BARKER, Ibid., idem, p. 33.

9. "About one third of the perforations in typhoid fever are associated with preceding hemorrhage, an association more common than you might suppose."—Ibid., idem, p. 35.

10. "We seldom see very high white cell counts in simple inflammatory processes within the abdomen. Marked leucocytosis (25,000 to 40,000) suggests very strongly that the blood supply to some abdominal viscus has been shut off."—LELAND MCKITTRICK, *New England J. Med.,* 205: 645, 1931.

Causes of Death

11. "Causes of death after 70: 1) arterial disease—heart, brain, kidney; 2) neoplasm; 3) terminal infection such as pneumonia and streptococcus disease; 4) prostatic obstruction and urinary sepsis. You will go right in 90 out of 100 cases if you make these assumptions."—R. CABOT, *New England J. Med.,* 205: 740, 1931.

12. "All testicular tumours should be considered malignant."—O. COX, *New England J. Med.,* 202: 243, 1930.

13. "Always do a pyelogram to be sure of a mass in the left upper quadrant—it may be kidney and not spleen, however sure you may feel on clinical examination."—ARLIE BOCK, *New England J. Med.,* 202: 1217, 1930.

14. "We see in patients (who are not getting enough fluid, who are passing a highly concentrated urine) albumin and once in a while, sugar that seems to be due to irritation of the kidney in secreting a highly concentrated urine."—ARLIE BOCK, Ibid., idem, p. 683.

ORIGINAL ARTICLES

The Selection of the Proper Operation for the Individual Ulcer Patient

By JOHN O. DAMPEER, JR., M.D., *Kermit, Texas*

In recent years it has been possible to perform definitive surgery for peptic ulcer, either gastric or duodenal, with a very low mortality and with extremely good assurance against recurrent ulceration. With this high "cure" rate, more attention has been focused in recent years on the post-gastrectomy symptoms and nutritional disturbances which have all too frequently been the price of the surgical ulcer cure.

What ever the more remote underlying cause, the final etiologic factor in peptic ulceration is acid-pepsin autodigestion of the patient's own tissues. This may be caused either by excess acidity or by reduced tissue resistance. The former is the usual mechanism in duodenal ulcer and the latter more frequent in gastric ulcer.

After it was realized that recurrent ulceration occurred with distressing frequency following gastrojejunostomy, subtotal gastrectomy became the standard operation for duodenal ulcer. This decreased the incidence of stomal ulceration to approximately three per cent.[5] Because this was not an entirely unmixed blessing, since undesirable after-effects were quite common, there has been a continued interest in the development of some procedure which would eliminate both evils.

Three Phases

In a considerable oversimplification, it might be said that there are three phases of gastric secretion. The cephalic phase, mediated by way of the vagus nerves, and the gastric phase, mediated by the hormone, gastrin, which is elaborated by stimulation of the antrum and travels by the blood stream to stimulate the parietal cells of the body and fundus, are the two most important. Although there is a complex interaction between these two phases, I believe that it is safe to state that the abolition of both eliminates approximately 90 per cent of the stimulation of gastric secretion. The remaining 10 per cent is chiefly the function of the intestional phase.[2]

Gastrojejunostomy combined with vagotomy, popularized chiefly by Dragstedt in this country, was the first departure which gained prominence. After several years it became evident that the incidence of recurrence was unduly high, in the neighborhood of 13-20 per cent.[5] For this reason this operation has been abandoned for the most part except in the occasional patient in whom it might be elected because of technical factors in the management of the duodenal stump or because of the patient's precarious general condition.

Gastroduodenostomy

The use of gastroduodenostomy by the Billroth I method in the reconstruction following excision of the stomach has been reevaluated by a good many surgeons. Most writers agree that the patients do better nutritionally and the incidence of the dumping syndrome is notably decreased. Unfortunately, however, the incidence of recurrence is increased in duodenal ulcer although it is not in gastric lesions.

In an interesting study conducted in Sweden, considering only those patients operated upon by two professors of surgery, it was concluded that the Billroth I operation is eminently satisfactory for women and for men with gastric ulcers. There was, however, a significant increase in the incidence of recurrence in men with duodenal ulcer, and they felt that this operation probably should not be used for the latter group.[7]

Limited Gastrectomy

In the past several years, a limited gastrectomy combined with subdiaphragmatic vagotomy has become increasingly popular. This operation is on very sound physiologic grounds, since it abolishes both the gastric and cephalic phases of gastric secretion.

Several large series of patients who have undergone this operation have now been followed for several years. The results have been very good. There are virtually no reported recurrences in the literature except for a few patients in whom it was established that the vagotomy had been incomplete.

There is general agreement among those surgeons who have experience with this operation that the patients do far better nutritionally, and there is the definite impression that the incidence of the dumping syndrome is markedly reduced.

Many people have adopted a "wait and see" attitude toward this operation, since they recall that for many years it was felt that simple gastrojejunostomy was quite satisfactory. If this operation stands the test of more time, it will probably be even more widely adopted.[3-6]

Stoma Size

For a long time it was felt that stoma size in the reconstruction of gastrointestinal continuity was of little importance. People felt that the functional stoma size was limited by the size of the jejunal lumen.

This is, of course, true for large stomas. However, recently there has been brought forth fairly convincing evidence that there is a definite relationship between the size of the stoma and dumping symptoms.

These writers showed that with stomas approaching the one centimeter internal diameter of the pylorus, both dumping symptoms and nutritional disturbances were greatly lessened. They admitted, however, that in the early post-operative period, some element of outlet obstruction was quite common with this small stoma.[1]

Considerable Choice

This leaves us with a considerable choice of operation for any individual patient. For gastric lesions, I believe a wise choice would be a Billroth I reconstruction except in the occasional patient with a high-lying ulcer in whom this is not feasible.

For the usual patient with duodenal ulcer, the conventional 70-75 per cent gastric resection of the Billroth II type probably should be the usual operation. I do feel that one should consider stoma size in the reconstruction and, although I would be hesitant to construct a one centimeter stoma, a fairly small one that will comfortably accommodate the thumb probably would be some assurance of a lowered chance of undesirable post-operative sequellae.

For those patients in whom nutrition is already a problem, and in men whose occupation requires strenuous physical labor, hemigastrectomy and vagotomy is probably the wiser choice. Whether the gastric resection is of the Billroth I or of the Billroth II type apparently makes little difference.

There are several well known methods for dealing with the difficult-to-manage duodenal stump, but rarely, particularly in the poor-risk patient, it is probably better surgical judgment to accept an increased risk of recurrence and to settle for a gastrojejunostomy and vagotomy.[4]

Summary

I. Several of the commonly used operations for peptic ulcer are discussed.

II. My own choice of operation in some of the commonly encountered situations is given.

110 N. Poplar Street

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Treatment of Arteriosclerosis
Intravenous Use of Allotropic Colloidal Iodine Solution

By Frederic Damrau, M.D., *New York*, and Albert B. Trencavel, Ph.D., *Los Angeles*

A clinical investigation of allotropic colloidal iodine solution*, administered intravenously, de monstrated that this medication is more effective in the treatment of arteriosclerosis than the same dosage of iodine given orally. In another series of cases intravenous injections of this solution were found to be free from any toxic reactions.

Oral Iodine

A survey of medical literature shows that the value of iodine and the iodides for arteriosclerosis has been known since 1876 (Wyckoff[1]) and is recognized by modern authorities.

In 1936 Damrau[2] reported favorable results following oral treatment of a series of cases of arteriosclerosis with a soluble iodine solution over a period of four months. In most cases there was effective relief of the symptoms of headache, dizziness, fatigue, palpitation and precordial pain. The author concluded that the benefit of this treatment "may be attributed to the fact that iodine acts specifically upon the disturbed cholesterol metabolism, which has been shown to be so important a factor in arteriosclerotic hypertension."

Sollmann[3] in 1957 confirms the present-day consensus of medical opinion that the iodides are useful in circulatory diseases. He writes: "Iodides are employed in arteriosclerosis, coronary sclerosis, angina pectoris, aortic aneurysm (Bouillaud, 1858), and the like. In the lighter grades of these conditions their continued administration seems to give gradual relief to the functional phenomena, especially the pain. The effects are not due to any change in the blood pressure, but probably to structural alterations in the vessel walls, perhaps absorption of cellular exudates."

Coronary Sclerosis

Keys[4] in 1957 emphasizes the importance of hypercholesteremia in the genesis of coronary sclerosis. He states: "It is well established that patients with coronary heart disease tend to have higher serum cholesterol concentrations than their clinically healthy counterparts . . . It seems that most middle-aged American men have hypercholesteremia in terms of the world distribution of serum cholesterol values. Autopsy studies indicate, further, that most middle-aged American men have severe coronary atherosclerosis. These facts are compatible with the theory that atherosclerosis is in considerable measure a reflection of the habitual serum cholesterol concentration.

"In American men, at least, coronary atherosclerosis is often well marked before they are 30 years old, so that the clinical appearance of the disease in men in the 50's represents the cumulative effects of many years."

In view of the established relationship between hypercholesteremia and arteriosclerosis, the value of iodides in reducing blood cholesterol is of especial interest.

Brown and Page[5] in 1952 reported that iodides inhibit the increase of cholesterol in the liver and blood and thus check the development of atherosclerosis. In 1954 Page[6] reaffirmed the traditional use of iodides in the treatment of arteriosclerosis and their value in combating cholesterol induced atherosclerosis in rabbits.

Cholesterol in Rabbits

Bruger and Oppenheim[7] in 1951 reported that the precipitation of cholesterol in the intima of the arteries, induced by feeding cholesterol to rabbits, is inhibited by iodides.

The increasing evidence of close relationship between hypercholesteremia and atherosclerosis, coronary sclerosis in particular, and the established value of the iodides in reducing blood cholesterol provide the rationale for using Colloidal Iodine Solution intravenously in the emergency and more effective treatment of arteriosclerosis and its sequelae.

Intravenous Iodine

As Goodman and Gilman[8] have said, "the iodides have been employed empirically in a wide

*Supplied as 5 cc. ampoules, also 30 and 60 cc. vials, of Iriodine Colloidal Iodine Solution by Trencavel Laboratories, P. O. Box No. 25853, Los Angeles 25, Calif.

variety of diseases too numerous even to be fully listed. As examples, one might cite their use in the treatment of encephalitis, essential hypertension, portal cirrhosis, thromboangiitis obliterans, etc."

Since the therapeutic indications for the iodides are so wide and diverse, it is not surprising that many papers have been published on intravenous iodine therapy.

Sodium Iodide Injection N.F. X[9] is an official drug for intravenous or intramuscular injection. It is classified as a **fibrillolytic.**

A survey of medical literature shows that intravenous iodine therapy has been reported by numerous investigators in a wide variety of diseases including (a) acute infections, (b) chronic infections, (c) degenerative conditions and (d) general disturbances.

Recent Reports

Of particular interest is a recent report by Miranda Ortiz and Gonzalez Calzada[10] on the palliative treatment of poliomyelitis with colloidal iodine solution. Favorable response to the medication was observed in 5 acute cases with full recovery after three weeks to three months. In a series of 65 chronic cases benefits of treatment were observed in all cases and included restoration of strength and tone in the affected muscles, improved local circulation, ability to stand and walk without aid, correction of contractures, and avoidance of the need for orthopedic surgery.

Bix[11] in 1925 reported the intravenous use of iodine for patients with atheromatous lesions in the blood vessels with definite relief of headache and dizziness. Leitloff[12] in 1928 observed relief of angina pectoris, as well as of headache and vertigo, following intravenous iodine. Other investigators who have reported benefit from intravenous administration of iodine in angina pectoris or arteriosclerosis include Lian, Blondel and Racine[13] in 1931; Jacobi [14] in 1937; Burlingame[15] in 1948; and Rosselli [16] in 1949.

Allotroic Colloidal Iodine Solution

This parenteral medication is a stable, neutral, isotonic and allotropic colloidal iodine solution supplied in 5 cc. ampoules, also 30 and 60 cc. vials. Each ampoule contains potassium iodide 10 mg., iodine 10 mg., beta amylose 100 mg. and distilled water q.s. ad 5 cc.

The recommended dose is 5 cc. by intramuscular or slow intravenous injection. In the treatment of arteriosclerosis it is administered twice weekly.

The unique features are the stability and allotropism of the element iodine in an aqueous colloidal solution dispersion preparation.

The ingredients of allotropic colloidal iodine solution, when mixed in accordance with the Trencavel process, form a stable isotonic and allotropic colloidal iodine solution having a neutral reaction of pH 7. It is essentially an aqueous solution of metalloid iodine, in potassium iodide as a solvent, suspended in an amylose colloidal medium. The iodine element is held peripherally to the complex amylose molecule by adsorption with an angle of rotation of approximately 159° (dextrorotatory).

Rationale of Vehicle

The rationale of the amylose vehicle is the fact that it holds the iodine metalloid by allotropism in two different forms, that is the physical and the chemical form, by adsorption and chemical solution. This is a unique characteristic not present in other colloidal iodine solutions. It is combined in this form for **gradual liberation of free iodine,** a condition best calculated for therapeutic purposes.

The allotropic characteristic provides a unique colloidal iodine solution in that the starch fraction is the specific antidote for iodine and renders it non-toxic and at the same time gives it the properties of a complex and harmonious amylaceous compound. The unusual therapeutic properties of allotropic colloidal iodine solution are due to this allotropism of iodine.

The iodine element is held firmly by adsorption with the amylose, to be released as free iodine gradually as needed on contact with the blood. The ampoules are tested for sterility and pyrogens in conformity with the requirements of the **U. S. National Formulary.**

Clinical Study of Safety

Forty intravenous injections of allotropic colloidal iodine solution were given to 20 patients under close observation to determine the presence or absence of toxic manifestations. Ten patients received injections on three successive days; the other 10, a single injection. In all cases the diagnosis was arteriosclerosis.

Sixteen of the patients were females and four males. The average age was 63, with a range of 49 to 73. The average height was 65 inches, with a range of 61 inches to 69 inches.

The technique of administration was slow intravenous injection, over a period of five minutes (1 cc. per minute), into the vein in the antecubital region. The pulse, temperature and blood pressure were checked before and after each injection.

Close observation was continued after each injection for the possible appearance of the following symptoms: (1) shock, (2) change of blood pressure, (3) fever, (4) tachycardia, (5) dyspnea, (6) headache, (7) vertigo, (8) nausea, (9) vomiting, and (10) other reactions.

None of the above reactions was observed in any case. One patient complained of slight tingling of the fingers during the first minute of the first injection. It disappeared when the needle was withdrawn and did not recur with two subsequent injections. Apparently it was due to fear of the procedure.

The conclusion was reached that slow intravenous injections of allotropic colloidal iodine solution do not result in toxic manifestations.

Therapeutic Results

Intravenous therapy with allotropic colloidal iodine solution, in comparison with the same dosage of potassium iodide and iodine administered orally, was evaluated in a series of 30 cases of diagnosed arteriosclerosis.

The series included 18 females and 12 males. The average age was 65, with a range of 50 to 78. The average weight was 153 lbs., with a range of 92 to 210 lbs. The average height was 65 inches, with a range of 60 inches to 73 inches. The average duration of the illness was 9.6 years, with a range of 4 to 20 years. Complicating hypertension (systolic blood pressure over 150 mm. Hg.) was present in 16 cases. Three cases were dropped from the study because of lack of cooperation.

The principal complaint was dizziness (17 cases), fatigue (16 cases), headache (12 cases), insomnia (8 cases), anorexia (7 cases), dyspnea (6 cases) and tinnitus (4 cases).

Intravenous injections of Iriodine were given twice weekly to all 27 patients for a period of two months.

27 Patients

As controls, the same 27 patients were given an oral medication containing the identical amounts of potassium iodide and iodine dissolved in water. This treatment was also continued for two months.

In 13 cases, the oral medication was given first and then changed to allotropic colloidal iodine solution after a rest period. In the other 14 cases the parenteral medication was administered first.

All 27 patients (100 per cent) showed clinical improvement while under treatment with allotropic colloidal iodine solution.

Dizziness, which was a complaint in 17 cases, was relieved under treatment with allotropic colloidal iodine solution in 13 cases (76 per cent). This symptom was relieved in 11 cases (65 per cent) under oral iodine medication.

Fatigue, which was a complaint in 16 cases, was relieved under treatment with allotropic colloidal iodine solution in 12 cases (75 per cent). This symptom was relieved in 8 cases (50 per cent) under oral iodine medication.

Headache, which was a complaint in 12 cases, was relieved under treatment with allotropic colloidal iodine solution in 9 cases (75 per cent). This symptom was relieved in 5 cases (42 per cent) under oral iodine medication.

Insomnia

Insomnia, which was a complaint in 8 cases, was relieved under treatment with allotropic colloidal iodine solution in 5 cases (63 per cent). This symptom was relieved in 3 cases (38 per cent) under oral iodine medication.

Anorexia, which was a complaint in 7 cases, was relieved under treatment with allotropic colloidal iodine solution in 6 cases (86 per cent). This symptom was relieved in 3 cases (43 per cent) under oral iodine medication.

Dyspnea, which was a complaint in 6 cases, was relieved under treatment with allotropic colloidal iodine solution in 5 cases (83 per cent). This symptom was relieved in 2 cases (33 per cent) under oral iodine medication.

Tinnitus, which was a complaint in 4 cases, was relieved under treatment with allotropic colloidal iodine solution in 2 cases (50 per cent). This symptom was relieved in 1 case (25 per cent) under oral iodine medication.

Case Report

P. C., male, age 67, weight 153 lbs., height 5'8", complained of dizziness, dyspnea and abdominal distress. These symptoms had persisted over a period of eight years. The peripheral arteries showed thickening with rubor of the extremities. The pulse rate was 78. The systolic blood pressure was 200 mm. Hg., diastolic 90. Routine laboratory tests were negative.

Slow intravenous injections of allotropic colloidal iodine solution were administered twice weekly for a period of two months. With this treatment all of the symptoms were relieved completely. The systolic blood pressure was reduced to 178 mm. Hg., diastolic 64. There were no untoward effects of the injections.

Summary

A comprehensive survey of medical literature shows that iodides have been used in the treatment of arteriosclerosis since 1876 and that their therapeutic value is recognized by modern medical authorities. It has been established that iodides reduce blood cholesterol and its deposition in the intima of the arterial walls, thereby checking the progress of atherosclerosis.

A clinical study has demonstrated that allotropic colloidal iodine solution, administered by slow intravenous injection, is safe and free from harmful reactions.

Intravenous injections of allotropic colloidal iodine solution twice weekly for a period of two months provided improvement in all 27 cases (100 per cent) of arteriosclerosis. Dizziness was relieved in 76 per cent of cases, headache in 75 per cent, fatigue in 75 per cent, anorexia in 86 per cent, dyspnea in 83 per cent, insomnia in 63 per cent, and tinnitus in 50 per cent.

Comparable benefits were not observed in a control study of the same patients orally for a similar period of two months with a solution containing the same amount of iodine and potassium iodide.

2 Tudor City Place.

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Oral Treatment For Ringworm Reported

An antibiotic, which can be taken by mouth, has been found to be effective in the treatment of ringworm, according to two Florida dermatologists.

In a preliminary report in the Archives of Dermatology, published by the American Medical Association, the doctors said they treated 31 patients with various types of ringworm with griseofulvin.

The drug is called griseofulvin for the species of penicillium from which it is made. Penicillin is made from another species — notatum. Griseofulvin earlier was found effective against fungus infections in plants.

When griseofulvin was taken daily by mouth, ringworm of the body and scalp cleared within one to three weeks. Onychomycosis (ringworm of the nails) cleared within three to four months, they said.

Adverse side effects from griseofulvin appear to be minimal in man, they said. However, animal experiments indicate that griseofulvin affects the blood and the doctors recommended that regular blood cell counts be made during prolonged use of the drug in humans.

Drs. Harvey Blank and Frank J. Roth of the University of Miami School of Medicine, Miami, Fla., and the Veterans Administration Hospital, Coral Gables, Fla., were the authors.

A Case of Chess Paralysis

This note on chess paralysis is written as a warning to those of the author's friends who are chess fiends rather than to add to the already cumbersome literature on the subject of neuritis.

As is well known, the diagnosis of neuritis is made too frequently and too loosely. Even if one limits the diagnosis to cases which meet definite criteria (of motor weakness, anatomically valid sensory change and tingling of the involved nerve), the causes of neuritis and neuropathy are legion. They may be classed as mononeuritis and polyneuritis due to refrigeration, compression, intoxication (heavy metals, Jamaica ginger, alcohol, etc.), infection, hypoxia and hypoxemia, poor nutrition, and so.

No photograph accompanies this paper. Any classic photograph in any standard text illustrating the effect of trauma of the ulnar nerve in the ulnar groove will suffice.

A young book salesman presented himself with classic right ulnar nerve paralysis of three weeks' duration. In reply to questions on a possible source of trauma to the elbow, the patient gave categorically negative answers. As he was leaving the office, he said, "You know, I began to learn chess four weeks ago. I rest my right elbow on the table and keep banging it there. Could this have anything to do with my trouble, Doctor?"

As someone is said to have said, "If you let the patient talk long enough, he will give you the diagnosis."

—Procrastines, *El Paso*

National Hospital Week, May 10-16

The nation's hospitals, through the American Hospital Association, have invited their closest ally, the medical profession, to help them develop greater understanding and appreciation of their services and contributions to the American people.

This year's program, centered in a seven-day observance of National Hospital Week, May 10-16, will emphasize the theme of "More Roads to Recovery." An explanation of these "roads"—better care, improved techniques and skills, greater numbers of personnel to apply the dramatic successes of medical science—will help offset a growing myth that hospital costs are greater than the services received.

Coming Meetings

New Mexico Medical Society, annual meeting, Las Cruces, N. Mex., May 5-7, 1959.

Annual Assembly, Nevada Academy of General Practice, Riverside Hotel, Reno, Nev., May 21-23, 1959. Program to be presented by the faculty of the University of Southern California School of Medicine.

The American College of Physicians, Postgraduate Course, Cardiac Arrhythmias, Philadelphia General Hospital, Philadelphia, May 22-24, 1959.

The American College of Physicians, Postgraduate Course, Special Topics in Internal Medicine, University of Colorado School of Medicine, Denver, June 15-19, 1959.

University of Colorado Medical Center, Fifth Annual Postgraduate Course, Dermatology for General Practitioners, Denver, July 16-18, 1959.

Rocky Mountain Cancer Conference, Brown Palace Hotel, Denver, July 22-23, 1959.

Nevada State Medical Association, Annual Meeting, Mapes Hotel, Reno, Aug. 19-22, 1959.

American Fracture Association, Annual Meeting, New Orleans, Nov. 1-3, 1959.

Southwest Obstetrical and Gynecological Society, Annual Meeting, El Mirador Hotel, Palm Springs, Calif., Nov. 2 and 3, 1959.

Southwestern Medical Association, Annual Meeting, Roswell, N. M., Nov. 5-7, 1959.

ATTEND THE
ANNUAL MEETING
OF THE NEW MEXICO
MEDICAL SOCIETY
IN LAS CRUCES
MAY 5-7

MONTHLY CLINICAL PATHOLOGICAL CONFERENCE
EL PASO GENERAL HOSPITAL

February 19, 1959

FREDERICK P. BORNSTEIN, *M.D., Editor—Case 1181*

Presentation of case by L. A. Gladstone, M.D.

History:—Dr. Nathan Kleban:

A 54-year-old Latin-American housewife entered the hospital Dec. 3, 1958. During the month before admission there had been a 20-30 lb. weight loss associated with poor appetite and occasional vomiting after eating. The patient had complained of pain in the left upper abdominal quadrant or left breast; "convulsions" had been occurring for three days. There was no observed blood loss.

No additional history was obtained.

Physical Examination:

While being admitted the patient had "an aborted grand mal" seizure not further described. T. 98.6, P. 84, R. 18, B. P. 160/100. She was obese and had icteric sclerae. Eye grounds were not visualized because of lenticular opacities. The abdomen was protruberant. One examiner recorded tenderness in the left upper quadrant, another in the right. There was uncertainty as to whether the liver edge was two finger-breadths below the right costal margin. The cervix was friable, eroded, bled easily; vaginal discharge was thick, white. Neurological examination was said to be within normal limits.

Hospital Course:

Spinal paracentesis was attempted after admission but was not successful because of the seizures. On the second day a pressure equivalent to 264 mm. of water was measured, dropping to 220 after 10 milliliters were removed.

Except for a mild cystocele and rectocele a repeat pelvic examination was considered within normal limits. Smears were made for Papanicolaou stain.

After the sigmoidoscope was introduced to 12 cm. the patient balked at further examination. A two by three cm. firm black fecal mass was encountered. There were external and internal hemorrhoids.

Treatment included whole blood, chloropromazine (Thorazine), phenobarbital, diphenylhydantoin sodium (dilantin), sulfamethoxypyridazine (kynex), iron, vitamins, calcium gluconate, chloramphenicol (chloromycetin) and high protein diet.

Seizures Not Described

Seizures (not described) stopped after the first day. She was later described as sluggish and mildly responsive. On the fifth day the patient sustained a hematoma of the forehead when she fell out of bed. Eleven days later spinal fluid pressure was not elevated and fluid was clear.

The patient improved to some extent, was able to get out of bed and go to the bathroom, was less confused.

Two ounces of castor oil and soap suds enema were given on the 13th and 14th days in preparation for sigmoidoscopy and barium enema. Confusion and weakness returned and increased. Headache developed. Stupor was followed by coma, convulsions and death on the 17th day, Dec. 19.

There was no fever during the patient's hospital stay. Diastolic blood pressure was usually below 90. The recorded urine output for 24 hours ranged from 1040 to 1250 milliliters. From the ninth to 15th days the patient went to the bathroom and urine output was not measured. There was no urine output during the last day of life.

Laboratory Findings:

X-rays: 12-4-58—Chest—"Sagittal view of the chest reveals no evidence of metastasis to the visualized bony thorax or lung fields. The heart and mediastinal structures appear natural. Conclusion: Negative for evidence of metastasis".

12-11-58—KUB—"Survey film of the abdomen reveals gas in the stomach and large bowel. Two opacities in the left upper quadrant are suggestive of possible renal calculi. The psoas shadows and

properitoneal fat lines are visualized bilaterally. Due to the habitus of the patient, the kidney shadows are poorly visualized. The lumbar spine and pelvis reveal no evidence of osteolytic or osteoblastic metastasis. Conclusion: Negative for evidence of metastatic disease; possible renal calculi on the left".

12-16-58—Barium enema—"Barium was instilled into the rectum and flowed easily throughout the colon to the cecum. There were no significant addition or subtraction defects. Evacuation film revealed good motor function. Conclusion: Negative for evidence of colitis or neoplasm".

12-17-58—Upper GI—"Examination of the upper gastro-intestinal tract reveals no evidence of ulcer or neoplasm. The esophagus is intact and there is a small hiatus hernia".

Electroencephalogram — 12-8-58 — "This record shows an absence of the normal rhythm replaced by diffuse generalized, moderately deep stupor suggesting a nutritional metabolic disturbance, including a possible hypoglycemia. Conclusions: Abnormal electroencephalogram, generalized diffuse stupor with increased pressure".

Electrocardiogram—12-18-58—"Electrolyte imbalance—(hyperkalemia and hypocalcemia)"

Vaginal and cervical Pap smears—12-5-58—negative for cancer cells.

Blood counts: 12-2-58—Hb. 6.5 gm. Ht. 19% WBC 12,400, Eosins. 4, Stabs. 1, Segs. 71, Lymphs. 20, Monos. 4, **12-8-58**—Hb. 11.1 gm. Ht. 32 vol.%, WBC 9,600, RBC 3.31, **12-16-58**—Hb. 7.8 gm. Ht. 28%. **12-18-58**—Hb. 13.3 gm. Ht. 40%.

Blood chemistry: 12-3-58—Alkaline phosphatase —3.6, Van den Bergh Direct—.077, indirect—.435, Glucose—140 mg.%, Urea Nitrogen—47. **12-4-58**—CO_2 combining power—20 mEq/L, Calcium—6.7 mEq/L, Sodium—168 mEq/L, Potassium—4.5 mEq/L, Chloride—132 mEq/L, Phosphorus—6.6 mg.%, **12-11-58**—Urea Nitrogen—87 mg.%, Calcium—5.7 mg.%, **12-15-58**—Urea Nitrogen — 99 mg.%, **12-17-58** — Calcium — 3.0 mg.%, Chlorides — 110 mEq/L, Potassium — 4.6 mEq/L, Sodium—128 mEq/L, **12-18-58**—Urea Nitrogen—101 mg.%, Potassium—5.2 mEq/L, Sodium—150 mEq/L.

Platelet count—12-8-58—125,780

Coagulation time 6 min., bleeding time 2½ minutes—12-8-58

Prothrombin time—100%—12-8-58

Spinal fluid—12-4-58—Total protein 19.5, WBC 2, polys 1, sugar 85, 15 rbc, **12-18-58**—Total protein 41, WBC 3, polys 1, Lymphs. 2, sugar 122, chlorides 150 mEq/L.

Bone marrow—12-11-58—1. Iron stains— moderately increased, 2. Red cell morphology—normal, 3. Routine studies—no significant changes.

Urine culture and sensitivity—12-2-58—Staph and coliforms—Sens., Chloro., Terra., aureo., neomycin.

Urinalyses: 12-3-58—(cath.) straw, clear, acid —S.G. 1.010, albumin 3+, sugar negative, WBC 8-12 (occ. cl.) RBC occ., occ. sq. ep. cell. **12-9-58** (cath.)—straw, cloudy, alkaline—S.G. 1.010—albumin 3+, sugar neg., WBC 1-2, RBC 2-3, many nonmotile bacteria, phosphate crystals, **12-9-58**—(cath.) yellow, turbid, alkaline, S.G. QNS, albumin 2+, sugar negative, WBC—rare, RBC 25-30, few sq. ep. cells **12-10-58**—(cath.) straw, clear, alkaline, S.G. QNS, albumin 2+, WBC rare, RBC rare, occ. sq. ep. cell, **12-11-58**—(cath.)—straw, clear, alkaline, S.G. 1.010—albumin 2+, sugar negative, WBC 2-3 with clumps, RBC 1-3, few sq. ep. cells, **12-14-58**—(cath.)—straw, clear, acid—S.G. 1.005—albumin 2+, sugar negative, 6-10 ep. cells, moderate bacteria, **12-16-58** —(cath.)—straw, cloudy, acid, S.G. 1.012—albumin 4+, sugar negative, WBC 18-20/HPF, rare squamous ep. cell, many motile bacteria, **12-17-58**—(cath.) straw, hazy, alkaline, S.G. 1.004—albumin 2+, sugar neg., WBC many large clumps, phosphate crystals, **12-17-58**—(voided)—clear, acid, S.G. QNS—sugar negative, innumearble WBC, rare RBC, **12-18-58**—straw, cloudy, acid—S.G. 1.007, albumin 3+, sugar negative, innumerable WBC.

Urine for bilirubin 12-9-58—negative.

Stool 12-15-58 showed traces of occult blood.

Clinical Discussion: Dr. L. A. Gladstone:

There is a saying that sometimes too much information can be just as confusing as too little information. Well, the CPC for tonight involves a 54-year-old woman whose total hospital stay was 17 days.

On the surface, as we examine the history, it appears as a straightforward case of an elderly patient who had lost weight, approximately 20 to 30 pounds over an undetermined period, who eventually went into renal decompensation and

died, with all the complications of renal insufficiency and uremia.

This patient apparently had an exhaustive study; there apparently are some very conspicuous omissions in the history. I don't know whether they were omitted purposely from the protocol or whether they were not done?

Dr. Nathan Kleban:

No. Nothing was omitted.

Dr. Gladstone:

I would like to see the X-rays at this point if I may, please.

X-Ray Discussion—Dr. William Melton:

I will limit the X-ray discussion to the positive findings. On the KUB there are some densities overlying the right renal region which have the appearance of large renal calculi. On the plain film on the right I can't identify the renal shadow; there is some motion on the film and the soft tissue structures are not well defined.

There is faint density over the right renal shadow that might raise the question of a stone; however, on the subsequent barium enema films the stones in the region of the left kidney are obvious and I don't see any densities in the region of the right kidney. The chest film showed no evidence of an active process or metastatic disease.

On the upper GI series a small hiatus hernia is demonstrated; beyond this I don't think the X-rays contribute anything.

Dr. Gladstone:

Thank you, Dr. Melton. We have a woman who was admitted to the hospital, who was unable to give any kind of a history. All we know is that she had lost weight, that she had poor appetite, that she had been vomiting, and that apparently she had some convulsions.

On neurological examination there is apparently no explanation for these convulsions. However, we do know that the patient was hypertensive, and that she also had a normal or upper limit of normal spinal fluid pressure, and that the spinal fluid essentially was of no help in coming to a diagnosis in this particular case.

She had no elevated protein, her spinal fluid chlorides were within normal limits. The spinal fluid chlorides are to a certain extent a reflection of serum chloride concentration in the blood, as are spinal fluid sugars or other electrolytes. It is not lowered as it might be expected in tuberculous meningitis for instance.

Significant Finding

About the only significant finding on physical examination was questionable icterus. However, if we examine the second page of the protocol we see that the patient did not have an elevated bilirubin. It is difficult to determine a borderline icterus clinically.

I would prefer to rely on the laboratory findings in this instance. The patient had lenticular opacities. Opacities bilaterally in a patient of 50 are not unusual. Causes, such as diabetes for instance, are to be considered, but we know that the blood glucose in this patient was normal.

Of other causes, such as parathyroid deficiency for instance, there is very little evidence. A primary parathyroid deficiency is an extremely rare disease; more commonly than not it is due to a surgical operation upon the thyroid.

A tumor or a hemorrhage into the parathyroid gland occasionally can destroy the parathyroid gland but there is no evidence for that. Radiation, various toxic drugs, intoxication such as dinitrophenol, anastoline, we have no history for that; so I assume that the lenticular opacities in this patient are in accordance with the patient's age and are due to degenerative changes in the lens.

Some Question

There was some question that this patient may have had some tenderness in the right or the left upper quadrant, we know that she had complained of left upper quadrant pain, it appears that apparently the patient had kidney stones, and I would assume that the pain in the left upper quadrant and possibly less severe tenderness was due to the stones in the kidney.

Numerous Examinations

The patient had a number of examinations. In a patient who loses 25 or 30 pounds obviously the possibility of a malignancy should be considered. The obvious sources of a malignancy such as the gastro-intestinal tract or the GU tract, I believe were pretty well excluded.

It is stated in the protocol that the patient had numerous pelvic examinations. I assume that they were done by competent people, and a malignancy such as carcinoma of the vagina which is a very

rare malignancy, or a carcinoma of the ovaries or of the uterus were not found.

Carcinoma of the colon: there is very little evidence for that. It is possible that the rectum may not be visualized in a barium enema. It was stated that the examiners had encountered some difficulty in examining the patient's rectum, and it is therefore conceivable that this patient may have had a rectal carcinoma with obstructive uropathy and a possible metastasis to the central nervous system and convulsions.

Usually one would expect to find some localizing signs and it is stated in this protocol that the neurological examination was normal.

Electroencephalogram

An electroencephalogram was done on this patient and as usual I believe there has been too much read into it. I don't think that one can be that positive on an electroencephalogram.

On an electroencephalogram I guess one can say that the patient has epilepsy or not, whether it is grand mal or petit mal, whether there is any absence of electrical activity over one hemisphere or the other or whether there are localizing spots, but I believe the gentleman who has read this electroencephalogram tried to be too cooperative.

The patient had bled. Bleeding tendency in an individual with renal insufficiency is not at all uncommon. What is the cause for it we do not know. There is such a thing as uremic colitis, uremic gastro-enteritis.

Uremia is a syndrome that involves practically every system in the body including the blood vessels; there is a lot of necrosis in the intestinal tract and bleeding. Also the serum calcium is lowered, that might be contributory to bleeding tendency.

There was no primary bleeding disease, I believe, because there was a normal coagulation time, normal platelet count, normal prothrombin time and also a normal bleeding time.

Treatment

The treatment at the beginning was apparently directed at the patient's symptoms. She had convulsions so she was given Dilantin and phenobarbital. It was thought, and proven, that she had renal infection; it was demontsrated on one whole half page of numerous urinalyses that she had had.

They all show a fixed specific gravity, albuminuria, pyuria and everything else that I believe goes along with a chronic renal insufficiency.

Therefore I assume that this patient's bleeding tendency was secondary to her uremia. She was also treated with intravenous fluids; there is no indication as to the rationale of this fluid therapy.

She received blood which is indicated in an individual who is bleeding and who is anemic. There is some question as to how much blood should be given to patients in uremia; we know they bleed.

You can give them a lot of blood but the patients do not keep it, for there is a certain element of hemolysis involved in uremia. Bringing up the hemoglobin or hematocrit to a reasonable level, let's say 10 to 11 grams, would be quite sufficient in an individual like this. She also was given isotonic sodium chlorides, and despite all this the patient's uremia progressed and so did her anemia.

Blood Urea of 47

On her admission she had, I believe, a blood urea of 47 which gradually increased and terminally it was in the neighborhood of about 100. Blood urea is a gross estimate of the patient's renal insufficiency. The degree of uremia is not at all indicative or a reflection of this patient's renal insufficiency or azotemia.

This might be why people prefer to call it azotemia rather than uremia. The patient had low calcium; there is a reason for that in a patient with uremia.

For one, it is one of the functions of the kidney to conserve calcium, number two, low calcium, hypocalcuria is a compensatory mechanism. The kidneys try to excrete fixed acids with any available anion that they have at their disposal: sodium, calcium, or magnesium, for that matter.

Patient Falls on Head

So what do we have here then? We have a patient who had lost weight, who had renal stones, who had become progressively uremic. During the course of the hospitalization she became somewhat less stuporous than she was, possibly secondary to correction of her internal environment by intravenous fluids. But then one day she fell on her head, that was approximately on the 14th day.

I am a little bit surprised that a patient who falls on her head and develops a hematoma does not have skull films. I believe they would have been very important. So we have a patient who has obviously renal insufficiency, who went into

uremia and who eventually died.

Cause of Uremia

Now, what does this boil down to? What is the cause of this uremia? Probably the most common cause of uremia in a patient with pyuria, fixed specifice gravity, is either a chronic glomerulonephritis or chronic pyelonephritis.

In a so-called end-stage kidney even the pathologist has a hard time to determine what it is. With pus in the kidneys I would assume this patient had probably chronic pyelonephritis. Or could this patient have had an obstructive uropathy?

Now what could cause obstructive uropathy in a female of 53? Probably a carcinoma arising from the pelvis, obstructing the ureters. Carcinoma of the cervix, a good proportion or a large proportion of patients with carcinoma of the cervix will die of uremia with obstructive uropathy, renal insufficiency.

Another Cause

Another cause of renal insufficiency in a patient of 50 is a congenital polycystic kidney. Of this condition there are two types: one, in children who die in their early infancy; the second is the adult type. Adults live to be 40 or 50 and usually there is a mass palpable, or masses palpable in both flanks but not necessarily so.

I have had the opportunity to see recently a patient who had died who had congenital polycystic kidneys; who was uremic; who had high blood pressure; and whose kidneys were not enlarged on a flat plate of the abdomen or by palpation by clinical examination.

Approximately one half of these people don't have hypertension; this patient was not hypertensive. They don't have to be hypertensive to be in renal insufficiency; the majority of them are.

Chronic Renal Insufficiency

So, to conclude, I would say that this individual probably did not have a malignancy causing her demise. Her uremia most likely was due to a chronic renal insufficiency secondary to chronic pyelonephritis or possibly congenital polycystic kidneys.

As far as her immediate cause of death, I do not know exactly but I would say this: this patient probably had sustained some sort of central nervous system injury, possibly an epidural or subdural hematoma, although she did not have elevated spinal fluid pressure.

In hematoma, the duration will depend on whether it is an arterial bleeding or whether it is a venous bleeding.

So I would say this patient probably had either chronic pyelonephritis uremia with renal failure or congenital polycystic kidneys and that probably the immediate cause of her demise was a central nervous system injury, possibly epidural or subdural hematoma.

Dr. H. M. Gibson:

I would like to make a remark or two about the interpretation of urine specimens from an indwelling catheter. The amount of pus that you find in a specimen, and to a certain extent the amount of blood that you find in a specimen, is utterly and completely unreliable.

In running urinalyses on people with indwelling catheters, you will find that 100 per cent of them have pus cells and frequently have some red blood cells. Generally you can depend on the specific gravity and the albumin and sugar of course in the urine, but please do not make a diagnosis of renal infection from indwelling Foley catheter.

The same applies after a patient is cystoscoped. We have seen people who have gone home or gone out of town and have been seen by their family doctor 4 or 5 days after they have been cystoscoped.

Then I get a call about midnight usually: this patient has albumin and blood in her urine. Well, of course she has, and 100% of them are going to have for about ten days. This patient obviously does have uremia and it does boil down to exactly why the patient has uremia.

I think most likely it is a chronic sclerosing pyelonephritis; the possibility of things like polycystic kidney disease are only that: possibilities. The evidence is certainly not very strong in support of them.

Possibility of Hyperparathyroidism

I would like to discuss the possibility of hyperparathyroidism in this case. Any person who has bilateral or multiple stones is a potential hyperparathyroid patient. The typical blood chemistry shows an elevated calcium and a lower serum phosphorous with increased calcium excretion in the urine.

However, in the presence of uremia these pic-

tures are frequently reversed. You may very well see the picture that you see right here, a lowered serum calcium and an elevated serum phosphorous, so this does not rule out a parathyroid disease.

The bones that we see here certainly have nothing that would make us think of a parathyroid disorder, but again, that is not reliable by a good percentage.

So I go along with Dr. Gladstone: I think this is a chronic sclerosing type of pyelonephritis, probably with stones, but I also consider the possibility that this patient may have parathyroid disease in addition.

Dr. Nathan Kleban:

This is in the nature of my usual carping comments. We had a patient several months ago in a similar situation, with far advanced, almost terminal renal insufficiency, who was given castor oil on several occasions followed by soap suds enemas and then went into stupor and coma and died within 24 hours.

This patient today and the one we had before, and I believe we had a third patient within the last year or so, certainly are people with far advanced disease. I think the people who have been taking care of them on the ward have not been mindful of the fact that they are in extremely precarious situation.

Anything which tends to deplete them severely of body fluid and lowering their circulating blood volume fairly rapidly, thereby lowering their renal blood-flow, can tip over an individual who is right on the verge of death from renal insufficiency.

Dr. Bornstein:

Assuming this patient had a sclerosing kidney disease, wasn't the blood pressure unusually low?

Dr. Gladstone:

Well, it is true that chronic renal insufficiency is frequently associated with high blood pressure, however, this is not necessarily so. There are certain types of renal insufficiencies that may not be associated with high blood pressure.

For instance, one is congenital polycystic kidney, another form is an amyloid kidney. Usually the amyloid kidney is the one that is described as the one with albuminuria without high blood pressure.

Dr. Bornstein:

What about chronic glomerulonephritis and chronic pyelonephritis?

Dr. Gladstone:

In chronic glomerulonephritis and chronic pyelonephritis, the patient usually has an elevated blood pressure. That blood pressure also depends to a certain extent on the state of the patient's hydration.

Dr. Kleban has brought out the possibility that this patient might have actually been depleted of body fluids.

A blood pressure of 160/100 can actually represent a shock in a patient who has previously been hypertensive but has been markedly dehydrated.

Dr. Postlewaite:

Grolman has done decapsulation procedures of the kidney, unilateral and bilateral, over a period of years. He has observed blood pressure changes and, under the stimulus of Tinsley Harrison, became interested in the question of whether there was a renin and antirenin factor in the kidney.

If you injure the circulatory system of the kidney, injure it by anoxia, then you get a renin or pressor effect. The unfortunate thing, however, is that nobody has taken time to discover what happened if there were no kidneys. In his recent book, Dr. Grolman reports the removal of both kidneys.

Keeping the dog alive for a period of several weeks by lavage procedures and electrolyte balance, he was able to maintain a completely normal pressor system within the animal. There was no evidence of hypertension or vascular changes secondary to it.

The dog was sacrificed; it could have been kept on except for limited factors of nutrition. I think there is evidence in the pyelonephritis group of diseases, if they are unilateral, that removal of this kidney will often give relief of the hypertension.

Dr. Bornstein:

I would like to make one more point. If you assume that this patient had a chronic glomerulonephritis or a pyelonephritis and is not hypertensive now, you would assume there had occurred a fairly recent drop in blood pressure. However, then you should have, at least roentgenologically, cardiac hypertrophy which you get after about a year of hypertension, and you would assume that this kidney disease is at least a year old, wouldn't

you? Dr. Melton, as far as the chest is concerned, is there any indication of cardiac hypertrophy on it?

Dr. Melton:

I didn't comment on it much, Fred, because I think that's a short distance film, but the heart looks enlarged to me, yes.

Clinical Diagnosis: Chronic pyelonephritis with renal failure.

Dr. Gladstone's Diagnosis: Either chronic pyelonephritis uremia with renal failure or, congenital polycystic kidneys.

Pathological Diagnosis: Polycystic kidneys, congenital

Pathological Discussion—Dr. F. P. Bornstein:

Autopsy revealed a poorly nourished 54 year old woman. The heart was not hypertrophied. The main findings, as was to be expected, were in the kidneys. (Fig. 1). This picture shows so-called congenital polycystic kidneys without renal enlargement. The right kidney was much smaller than the left and totally non-functioning. The left kidney was not enlarged but also completely cystic. The discussion brought up the question of a stone.

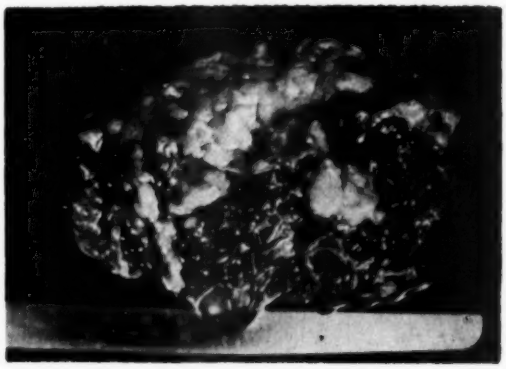

Figure 1

We did not find any real stone, only soft necrotic material with some calcified tissue in the center. On cut surface one sees that the renal parenchyma has been replaced by a conglomeration of cystic structures suggesting the complete absence of functioning renal tissue. This is a little bit deceiving, because some renal tissue must have been left, otherwise the patient wouldn't have lived to the age of 54.

Microscopic examination showed an agglomeration of lesions; namely, the basic polycystic lesion and a superimposed arterial nephrosclerosis.

Head Essentially Negative

The head was essentially negative so I think that uremia and the kidney failure are responsible for the patient's death. There is very little else to comment on it. It has been baffling to me why the woman lived for such a long time and lived fairly asymptomatically. What suddenly happened to destroy the kidney that quickly?

As to the development of this lesion, the favorite theory is that the kidney embryologically is composed of two parts which grow towards each other and intermingle. If this intermingling does not take place, or is prevented, a polycystic kidney is produced.

Attractive Theory

It is an attractive theory but I am not too happy about it because the last* Archives of Pathology reported a case of a space-occuying lesion in the brain. A mass was found which was composed of adrenal gland tissue which was checked by Drs. Klemperer and Mostafi and several other pathologists.

The adrenal gland, just like the kidney, supposedly has two different "anlage" which come together and form it.

Here you find both parts perfectly developed. So, whether or not one part has enough organizing ability to produce the other, or whether you really have to postulate this coming together from two directions, we don't know yet.

I want to say that we cannot say that the fusion theory is absolutely valid, because here we have an example that two parts can develop within a completely different organ if some primary malformation occurs.

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*Vol. 67, 1959—P. 228—"Intracranial adrenal Gland—Dr. Morris Wiener and S. Alton Dallgaard.

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